UNITED STATES DISTRICT COURT
DISTRICT COURT OF NEVADA

LaKISHA NEAL-LOMAX, JOSHUA WILLIAM LOMAX, ALIAYA TIERRAEE LOMAX, JUANITA CARR, as parent and guardian of INIQUE ALAZYA LOMAX, and JOYCE CHARLESTON, individually, and as Special Administrator of the Estate of WILLIAM D. LOMAX, JR.,	)
Plaintiffs,	)
vs.	) Case No.CV-S-05-01464-PMP-RJJ
LAS VEGAS METROPOLITAN POLICE DEPARTMENT; OFFICER REGGIE RADER, in his individual and official capacity; SHERIFF BILL YOUNG, in his official capacity; TASER INTERNATIONAL, INC., an Arizona Corporation; TASER INTERNATIONAL, INC., a Delaware Foreign Corporation; DOES I through X; DOES XI through XX; and ROE CORPORATIONS XXI Through XXX, inclusive,	)
Defendants.	)

Expert Report: Donald M. Dawes, M.D.

Pursuant to Fed. R. Civ. P. 26(a)(2), I, Donald M. Dawes, hereby submit my report that contains a complete statement of all opinions to be expressed and the bases and reasons therefore; the data and other information I considered in forming the opinions; the exhibits or list of references I used as a summary of or support for the opinions; my qualifications, including a list of all publications authored within the preceding ten years; the compensation to be paid for the study and testimony; and a listing of any other cases in which I have testified as an expert at trial or by deposition within the preceding four years.

Donald M. Dawes

April 16, 2007
Date

Lakisha Neal-Lomax v. Las Vegas Metropolitan Police Department, TASER International, Inc., et al. (A513602)

Expert Opinion of Donald M. Dawes, M.D.

Qualifications

I am an ABEM board-certified emergency physician practicing in a community hospital in Lompoc, CA. I am also an external medical consultant to TASER International, Inc., and have conducted numerous studies on the human effects of conducted electrical weapons. I am a sworn reserve peace officer in CA and authorized by my department to carry a TASER® X26 for duty having completed the required training course. My educational background includes a B.S. in electrical engineering from Cornell University, and an M.D. from Duke University. I completed my residency in emergency medicine at the Harbor-UCLA program in Torrance, CA. In addition to research and publishing on the human effects of conducted electrical weapons, I have also been an invited speaker to present on the subject of in-custody death. I have separately supplied my CV which includes a list of publications. I have also separately supplied my fees for expert testimony. I have no prior expert testimony experience.

Exhibits

Much of the research presented in this opinion is original and in various stages of analysis and publication. This data can only be released under a protective order in order to preserve its content for publication later under the peer-review process.

Facts of the Case

These are the facts of the case by my reading of the supplied documentation:

1) Security officers and the LVPD officer found Mr. Lomax in a severely compromised state that required urgent medical evaluation. Observations

supporting this included: a statement by a security officer that he "did not look good", a statement that he was "so out of it that we were actually worried about him", that he had an odor familiar to security officers as being consistent with "sherm" (PCP) ingestion, that he was walking in circles, was swaying back and forth, was lifting his shirt up and sweating profusely (though the ambient temperature was reportedly cool), was clenching his teeth and fists, was tensing up, was breathing hard, was responding not at all or inappropriately, was "glassy-eyed", was observed to be banging his head, stared through security officers and the LVPD officer attempting to communicate with him, was grabbing his abdomen and bending over, attacked a security officer unprovoked, had "incredible strength", and was noted by one security officer to have a fast heart rate (also noted by EMS on their initial evaluation later). By EMS statements, he required restraint for their evaluation and transport. Security officers and the LVPD appropriately activated EMS early.

2) Mr. Lomax had several medical co-morbid conditions at the time of his death which included: a history of chronic drug abuse, obesity, cardiac foci of interstitial fibrosis on autopsy, evidence of hepatitis on autopsy, and evidence of pneumonia on autopsy.

3) Mr. Lomax committed several crimes for which detention was appropriate: trespass, under the influence of a controlled substance, and battery. In addition, he had a prior history of assaultive behavior while under the influence. And PCP, which he was suspected to have abused, specifically is well known for inducing violence and volatile and unpredictable behavior. Most PCP deaths have been related to behavior while intoxicated. He posed a danger to himself and the public. One security officer testified that he was specifically concerned that Mr. Lomax could hurt someone else or himself if they just let him leave.

4) Mr. Lomax had acutely ingested PCP (phencyclidine) and likely marijuana (the combination of the two can be smoked – street names include "killer joints" and "crystal supergrass"). There are drugs of abuse that are not readily detected by usual screening as well. "Sherm", the street name for PCP, has been commonly combined with cocaine and embalming fluid, the latter not on standard screening tests. Embalming fluid can induce neuropsychiatric effects as well as slows the burning process during smoking.

5) The security officers and the LVPD officer specifically requested EMS transport for a medical evaluation. EMS (Fire) admitted initially delaying intervention since Mr. Lomax was not completely controlled despite manual restraint with several security officers and handcuffs. The Fire Captain testified that he ordered his men to stay back until the subject "calmed down." In addition, the LVPD officer was required by EMS to change the restraints for transport, lengthening the time of struggle. The Fire Captain testified that "the officer looked at us like for us to do something, and I basically told them we couldn't do anything while he was still in hard restraints."

6) Scene safety with the threatening crowd placed more time pressure on the security officers and the LVPD officer to obtain control and to effect transport. It caused EMS to move the patient without a complete evaluation and optimal patient care positioning. The AMR medic testified that the crowd was "diverting everybody's attention from the patient."

7) No other use of force was used other than manual restraint (including some compressive force with a knee to the sub-scapular region by a security officer), handcuffs (2 linked together), the TASER X26 used in the drive-stun mode, and the medical "soft" restraints. The time duration of the knee on the back was reported to be about 5 minutes. Other use of force options at the disposal of the LVPD officer included the expandable baton, oleoresin capsicum (pepper spray), and the lateral vascular neck restraint. None of these were used by the LVPD officer.

8) Mr. Lomax was placed prone for the restraint. However, according to deposition statements, his airway was not compromised by the covering of his mouth or excessive neck positioning. His head was either held with his face to the side or was freely mobile. Several deposition statements remarked on the free movement of his head with attempts to bite the restraining security officers. He was not placed in a "hobble" restraint. Additionally, two handcuffs were used (linked together) to restrain his hands behind his back, rather than just one to accommodate his large size (to not hurt his arms per the testimony of one security officer). The exact method of restraint on the AMR gurney was not clear by the testimony, but the testimony indicated that he had a waist belt in addition to the extremity restraints.

9) The TASER X26 was used in the drive-stun mode 7 times in 9 minutes and 55 seconds for a total duration of 31 seconds in two groupings. The shortest exposure was 2 seconds, and the longest exposure was 8 seconds. It is important to note that the downloaded times may not correlate to exposure to Mr. Lomax since it measures discharge, not contact. These would be the maximum exposures. The first two exposures were given 13 seconds apart. The last 5 exposures (begun 7 minutes and 42 seconds after the start of the last exposure from the first grouping) were given 13 seconds, 46 seconds, 33 seconds, and 28 seconds apart. The last exposure was not the longest exposure. The TASER was applied to the left posterior neck by both deposition statements and autopsy photographs. Autopsy photographs show superficial burn marks consistent with a TASER X26 used in the drive-stun mode just below the angle of the mandible beginning at about the posterior pinna and extending dorsally. This is an area heavily invested with neck musculature including the trapezius, the levator scapulae, the semispinalis capitus, the splenius capitus, the longissimus capitus, the sternocleidomastoid, and the deep cervical muscles. It is anatomically distant from the airway and the major nerves and blood vessels of the neck. The autopsy report revealed no injuries to the soft tissues of the anterior neck. See Addendum 3 for anatomic references.

10) The incident summary provided demonstrates that the last TASER X26 exposure was at 17:52:30. This was before Mr. Lomax was transferred to the ambulance. There is a notation that an IV was established in the right upper extremity and Mr. Lomax was still breathing at 18:25, over 30 minutes after the last TASER exposure. This timeline seems inconsistent with testimony and needs further clarification (most of the testimony seems to suggest some time on the gurney getting restrained, a half-minute to one minute transport to the ambulance, and several minutes of initial care in the ambulance before arrest). Both the AMR and Fire medics testified that Mr. Lomax was still breathing and resisting in some manner on transport to and in the ambulance, although he was possibly less agitated and seemed less responsive (specifically less verbal). The AMR medic testified that Mr. Lomax was trying to "rock off the gurney" on transport to the ambulance. The AMR medic also testified that Mr. Lomax resisted the tourniquet application for the IV start in the ambulance and was "grunting". A Fire medic did check the pulse after the last TASER exposure and it was present and estimated to be > 100. A Fire medic also checked his breathing by listening and by feeling his thorax movements and felt his breathing was adequate. The

LVPD officer also testified that he saw Mr. Lomax breathing on transport to the ambulance, noting his thorax was rising and falling.

11) The use of the TASER was in accordance to LVPD use of force policy according to testimony, and was only used to obtain compliance for manual restraint. Specifically, this was initially required for handcuffing (2 times) and then for changing the restraints from handcuffs to the "soft" restraints required by EMS (5 times). At both times, Mr. Lomax was resisting the restraint process according to testimony. The TASER X26 exposure allowed temporary incapacitation to perform the restraint function in the opinion of the LVPD officer and other testimony.

12) Mr. Lomax was noted to be bleeding from his mouth and to be biting on his own tongue on transport to the ambulance. This was not during the application of the TASER X26. This may have compromised his airway. At autopsy, mild tongue trauma was found.

13) Mr. Lomax was turned over after the initial medical care in the ambulance (length of time variably reported, range 1-5 minutes). To perform the functions documented, including an IV start with some resistance, the insertion of the nasal airway, and then recruitment of assistance to roll Mr. Lomax over probably would favor the longer timeline. At the time he was rolled over, he was noted to not be breathing and had no pulse. His initial rhythm was asystole, not ventricular fibrillation.

14) Mr. Lomax had at least 40 minutes of ACLS in the emergency department until the return to spontaneous circulation.

15) Mr. Lomax had, among other things, severe rhabdomyolysis. This is a testimony to the severe exertion he incurred during the struggle. His initial CK in the emergency department was 1917, and it rose to 128205 in less than 24 hours. Note that PCP use has been associated with rhabdomyolysis in the medical literature.

16) Mr. Lomax was severely acidotic at the hospital. He arrived at 18:30 and had spontaneous circulation by 18:55. The incident summary suggests a 6-minute transport time and transport was initiated almost immediately on determining he was in asystole per deposition statements. His first pH was < 6.5 at 19:16. His pH at 19:59, about an hour after the return to spontaneous

circulation and with an intravenous bicarbonate drip, was recorded to be 6.989.

17) Mr. Lomax had hyperkalemia on first blood draw (6.0 at 20:00).

Contributions to the Death of Mr. Lomax

In my opinion, with a reasonable degree of medical certainty, the following factors contributed to the death of Mr. Lomax:

PCP Toxicity

PCP affects a number of neurotransmitter systems including acting as a dopaminergic agonist (PCP is used as a model of schizophrenia in animals), complex interactions with the nicotinic and muscarinic cholinergic systems, acting as an NMDA antagonist, and poorly understood interactions with the serotonergic and noradrenergic systems. These complicated interactions make predicting effects difficult. A review of clinical findings in 1,000 cases of PCP intoxication (McCarron, et al. Ann Emerg Med, 10:5, 1981) showed that the following can occur: hypertension (57%), acute brain syndrome (37%), unconsciousness (11%), lethargy or stupor (7%), violent (35%), agitated (34%), and bizarre (29%) behavior, hallucinations or delusions (19%), mutism or staring (12%), nudism (3%), generalized muscle rigidity (5%), grand mal seizures (3%), profuse diaphoresis (4%), bronchospasm (2%), tachycardia (30%), apnea or respiratory arrest (3%), hyperthermia (3%), cardiac arrest (0.3%), hypotension (2%). The drug can cause increased cardiac inotropy and chronotropy or, instead, depression of myocardial contractility depending on the dose. It also causes a dissociative state and can, probably through its interaction with NMDA receptors, cause anesthetic effects. This may explain why subjects intoxicated with PCP have been reported to have extreme strength. Hyperthermia, though less common in PCP intoxication, is almost invariable fatal. This acute PCP intoxication (with possibly marijuana and other, undetected, drugs) may have been the etiology of the initial medical emergency in Mr. Lomax or it may have precipitated and contributed to another emergency medical condition such as the excited delirium syndrome (discussed below).

The post-mortem drug level of PCP was 129 ng/mL in Mr. Lomax. Though less than the reported lethal dose of PCP, this level is well within the

toxic range for this drug and is a level that can cause death from the direct effects of the drug. The fact that his drug levels continued to rise between an initial sample (28 ng/mL), and one done post-mortem (129 ng/mL) supports an acute ingestion of the drug (also supported by his odor, acute ingestion history per a deposed family member, and his behavior). The 129 ng/mL may also not have represented the peak level. It is also important to note that the correlation between body fluid levels and brain receptor binding of PCP is poor. This fact and individual variation in sensitivity to the drug makes correlation of drug levels and effects not particularly reliable. Some studies have shown brain changes similar to those seen in schizophrenia with chronic abuse. Mr. Lomax was known to have abused this drug previously (also associated with violence and also previously requiring medical intervention). Additionally, the manufacture of PCP involves many dangerous chemicals, and the drug can be contaminated with these dangerous chemicals significantly increasing its danger for lethality. Furthermore, there are many PCP analogue compounds which have unknown other side effects. Also, as previously mentioned, PCP can be combined with other dangerous compounds such as embalming fluid. Embalming fluid was not on the toxicology screen of the coroner. Embalming fluid can cause many dangerous effects including respiratory, metabolic, and brain function effects.

Excited Delirium

Excited delirium is a syndrome that is characterized by extreme agitation, delirium, hyperthermia, sweating (or, dry hot skin), undressing, attraction to lights and glass, violence towards inanimate objects, incoherent shouting, unusual strength, imperviousness to pain, and continued struggle even when confronted with overwhelming force with its etiology attributed possibly to chronic illicit drug abuse or chronic psychiatric illness, specifically schizophrenia and mania. This is felt to possibly be a distinct syndrome from the acute drug intoxication syndromes with stimulants such as cocaine, MDMA, methamphetamine, and PCP, although there is much overlap in these syndromes, and acute abuse of illicit drugs is often found in excited delirium cases (although not infrequently in lower than expected levels). Dr. Mash at the University of Miami has found distinct dopamine receptor differences between normal control subjects, cocaine intoxicated subjects (chronic users), and excited delirium subjects. She has found an expected compensatory increase in the dopamine transporters in the chronic cocaine users, but a lack of this compensatory brain change in excited

delirium subjects. In addition, the transporters present in the excited delirium subjects appear to be dysfunctional. She has hypothesized based on this that in acute cocaine abuse in these patients, because of the lack of the compensatory increase in dopamine transporters and their dysfunction, largely increased amounts of dopamine are present in the nerve synapses leading to the clinical manifestations in the excited delirium syndrome. Whether this is a maladaptive change to chronic drug abuse or a genetic susceptibility has yet to be elucidated. But this does point to a different etiology of the syndrome than just acute drug intoxication.

The excited delirium syndrome is not a new phenomenon and has been reported in the medical literature since the mid-1800's (referred to as acute exhaustive mania, Bell's mania, psychotic furors). It appears to be consistently associated with numerous in-custody deaths. Between 1915 and 1937, there were 360 cases in which the cause of death was listed as "exhaustion due to mental excitement" in one South Carolina hospital. There appeared to be a decrease in the reporting of this syndrome with the advent of anti-psychotic medications (e.g., chlorpromazine) in the 1960's, and a resurgence in the 1980's with the widespread abuse of cocaine (particularly "crack") and with the increase in the mentally ill released into the community (deinstitutionalization). A more recent resurgence may be due to the widespread abuse of methamphetamine. A hypersympathetic state, hyperthermia, electrolyte disturbances (particularly potassium), and a profound metabolic acidosis have been suggested as possible final common pathways in the respiratory and cardiovascular collapse in the excited delirium syndrome.

In Stratton, et al. ("Factors Associated with Sudden Death of Individuals Requiring Restraint for Excited Delirium", Am J Emerg Med, 19:3, 2001), the demographics of their excited delirium victims were described and Mr. Lomax had similar characteristics. In the Stratton, et al. series, 78% tested positive for stimulant drug use, 45% had chronic drug abuse by history, 56% had autopsy evidence of a chronic disease state, and 56% had obesity. Mr. Lomax tested positive for PCP. He had a toxic level, although not lethal level, of PCP in his system (we already established the poor correlation between levels and effect). It is not uncommon for excited delirium victims to have non-lethal levels of drugs in their systems at the time of death. As mentioned above, this may be due to brain changes associated with chronic drug abuse. Mr. Lomax was a chronic drug abuser by history. Chronic abuse of PCP can cause long-term brain changes similar

to schizophrenia (it is used as a model for schizophrenia in animals). Dr. Mash has also theorized chronic drug abuse may create brain changes that may be the etiology of the excited delirium syndrome. Her research has focused on the changes in dopamine receptors in the brain in excited delirium victims. Mr. Lomax was also found to have some evidence of chronic heart damage at autopsy (interstitial fibrosis or scarring). He also had obesity, with a BMI > 30.

Mr. Lomax had many of the classic behavioral patterns of the excited delirium syndrome. He exhibited bizarre behavior including walking in circles, swaying back and forth, clenching his teeth and fists, tensing up his muscles, responding inappropriately to security officers or not at all, and attacking one security officer without provocation. He was sweating profusely and lifting his shirt up even in cool weather (possibly hyperthermic), was "glassy-eyed", and had "incredible strength." Furthermore, in excited delirium cases there is a classic quiescent period immediately prior to sudden death. This is also described in the Stratton, et al paper. In the Stratton, et al series, the "cessation of struggle against restraints and onset of shallow or labored breathing is a sign that was described in each of the sudden deaths." It is additionally noted that "without exception, all cardiopulmonary arrests were unanticipated and preceded by a short period (estimates 5 minutes or less) during which the victim ceased in struggling against restraints and developed a labored or shallow breathing pattern." We know Mr. Lomax became less combative after the last restraint process (changing from handcuffs to "soft" restraints). This was likely his quiescent period. The fact that this occurs without TASER use (in the Stratton et al series, only 28% had a TASER used) means this is less likely a result of the TASER, and most likely a terminal phase of the excited delirium syndrome.

Acidosis is felt to be one of the common mechanisms of death in the excited delirium syndrome. Mr. Lomax was profoundly acidotic on initial blood draw (pH < 6.5) about 7 minutes after the return of spontaneous circulation. How much of this is related to his cardiac arrest and how much led up to his arrest is debatable, but given the entire clinical picture, I believe this derangement began in the field. He had a witnessed arrest with almost immediate ACLS by EMS and a very short transport time to the ED. His level of acidosis, in my opinion, was extreme given this almost immediate ACLS and given that the treatment in the ED consisted of bicarbonate therapy even prior to the first blood gas. The acidosis likely preceded his

arrest. Exertion alone can cause an acidosis (by the creation of lactic acid). As commented on by Hicks et al ("Metabolic Acidosis In Restraint-Associated Cardiac Arrest: A Case Series", Acad Emerg Med, 6:3, 1999), exertion coupled with a sympathomimetic drug (such as PCP) can lead to severe acidosis. If psychosis or delirium is present, especially when nocioception (pain sensation) is impaired, this can lead to even a more profound acidosis. The consequences of acidosis are systemic and include decreased cardiac contractility, arteriolar dilatation and venous constriction leading to central congestion, increased pulmonary vascular resistance, decreased cardiac output, sensitization to cardiac arrhythmias, decreased respiratory muscle strength and hypoventilation, hyperkalemia, protein degradation, loss of intracellular water regulation, obtundation, coma, and death.

One plaintiff expert opinion states that Mr. Lomax's degree of acidosis in the emergency department is "most consistent" with a primary respiratory acidosis. Relating his blood gas results at 19:16 to his arrest between 18:25 and 18:29 (by the incident summary) is difficult. However, given his extreme rhadomyolysis (discussed below), there is almost certainly a substantial contribution from a lactic acidosis. In addition, in a pure respiratory acidosis, the serum bicarbonate generally will rise, not fall. His bicarbonate was very low (< 7 on initial blood draw) and therefore, in my opinion, his low pH had to include a substantial metabolic acidosis. One of the admitting consultants also listed on the admitting diagnosis "profound metabolic acidosis." There is a theory that the primary arrest in the excited delirium syndrome is respiratory, rather than cardiac. One theory revolves around the catecholamine persistence during the quiescent period. Catecholamines drive potassium, released from actively contracting muscles, back into the cells. This is thought to be a protective measure to prevent hyperkalemia. Once the muscle contraction ceases, the catecholamines are still circulating at high levels for a brief period. This leads to a brief period of hypokalemia. In normal hosts, this is if no consequence. However, in persons already severely compromised, such as in an excited delirium or drug toxicity, this may be of great clinical significance. This period after exercise has been referred to as the "period of peril." Hypokalemia can cause respiratory arrest independent of cardiac arrest. Additionally, acidosis itself can lead to primary respiratory arrest as the muscles of respiration are significantly weakened leading to hypoventilation and brain function, to possibly include medullary control of respiration, is severely compromised.

Hyperthermia is also felt to be one of the common mechanisms of death in the excited delirium syndrome. Hyperthermia can cause systemic dysfunction. For example, in exertional heat stroke, mental status changes often are the first warning signs. This can be manifest by irritability and aggressiveness to bizarre behavior and psychosis to delirium and coma. Autonomic instability, lactic acidosis, rhabdomyolysis, hypo- or hyperkalemia, acute renal failure, DIC, and cardiovascular or respiratory collapse can occur. Mortality correlates with the severity and duration of the hyperthermia. The only notation I find about his initial temperature is in an admitting physician note ("afebrile"). It is not clear whether his temperature was taken during the resuscitation or how long after his return to spontaneous circulation (there was no note of his temperature on the resuscitation flow sheet and this is often a vital sign omitted from arrest victims by my experience). There is a notation in the chart later (2/21) of a temperature of >101. He did have undressing behavior and sweating in the field. Whether hyperthermia was a factor is debatable.

Finally, in the Stratton, et al. case series of excited delirium deaths, ventricular fibrillation was seen in none of the victims. The unusual aspect of these deaths is that the majority of the primary arrest rhythms were asystole. This is a common finding in deaths due to excited delirium, and, more importantly, generally not considered an arrest rhythm from electrical injury to the heart (unless extreme currents are used, such as with a lightning strike) and generally not the arrest rhythm found in sudden cardiac deaths in the younger population. Asystole was the terminal rhythm of Mr. Lomax. Profound acidosis, hyper or hypokalemia, and hypoxia can lead to this terminal rhythm.

In summary, Mr. Lomax had many classic signs, symptoms, and a course that was similar to deaths from the excited delirium syndrome. It is my medical opinion that he had this syndrome.

The Question of the TASER Association

There is some question as to whether or not the 31 seconds of the TASER X26 contributed to the death of Mr. Lomax. The following is a review of the current literature on the TASER as it may relate to this case:

Cardiac Effects

Given the drive stun use and the location of the use, and given that the arrest was asystolic (not commonly associated with electrically induced cardiac arrests), there is not much debate about whether the TASER X26 caused a malignant arrhythmia or other cardiac affect (except perhaps as related to a stress response or bradycardia, both discussed below). There are many studies that have failed to show a cardiac effect even in thoracic exposures. Some of these studies are listed in Addendum 2 for reference.

Rhabdomyolysis

We know Mr. Lomax had severe rhabdomyolysis. This can result from drug toxicity directly and extreme exertion. Ho, et al. did the first published study on the effects of the TASER X26 on humans ("Cardiovascular and Physiologic Effects of Conducted Electrical Weapon Discharge in Resting Adults", Acad Emerg Med, 13, 2006). In this study, 66 subjects were exposed to a 5-second discharge from a TASER X26. Serum chemistries were analyzed serially (up to 24 hours). The mean CK was found to rise slightly from 185 to 242. In this study, the exposure was to the heavily muscled back with roughly 12-18 inches of spread between the probes (the TASER was fired from about 7-10 feet). This rise, while statistically significant in the study, was certainly of no clinical significance. In this case, Mr. Lomax had the TASER applied to the musculature of the posterior neck. This is a small muscle mass compared to the area targeted by the Ho, et al. study and the TASER was used in a drive-stun mode which is much poorer at muscle recruitment both because of the surface application and the small spread between the probes. Mr. Lomax had significantly higher CK levels than the Ho, et al series (1917 initially, rising to 128205 in less than 24 hours). The TASER X26 likely had no role in this elevation. This elevation was likely the result of his PCP intoxication, his excited delirium syndrome, and the severe struggle under restraint.

Breathing

In our study published in Academic Emergency Medicine (Ho, et al., "Respiratory Effect of Prolonged Electrical Weapon on Human Volunteers", Acad Emerg Med, in press, 2007), we found that a 15-second exposure of the TASER X26 across the diaphragm (abdomen to upper thorax) did not impair the measured respiratory variables. In fact, tidal volume, respiratory rate, and end-tidal oxygen increased and end-tidal carbon dioxide decreased during the 15-second exposure. One plaintiff

expert opinion speculated that the TASER X26 might prevent the compensatory increase in ventilation necessary to offset a lowered blood pH. Our study does not support this theory since our study subjects were able to increase their ventilation during the exposure.

One plaintiff expert opinion referenced the Jauchem, et al. study (Jauchem J, Sherry C, Fines D, and M Cook, "Acidosis, lactate, electrolytes, muscle enzymes, and other factors in the blood of sus scrofa following repeated TASER exposures, Forensic Sci Int, in press, 2005). This study used a swine model. The swine were exposed to the TASER X26 as 5 seconds on then 5 seconds off for 3 minutes straight. This means 1 ½ minutes of exposure in 3 minutes with only 5 second breaks between. Mr. Lomax, in the second group of exposures, only received 22 seconds of exposure in 1 minute and 47 seconds with breaks of 13 seconds, 46 seconds, 33 seconds, and 28 seconds. The Jauchem, et al. study was a 50% duty cycle compared to the 20% duty cycle that Mr. Lomax was exposed to. Additionally, the swine had the TASER exposures trans-thoracically (chest to abdomen) with the probes piercing the skin. Mr. Lomax only had a drive-stun application to the neck. This is a smaller muscle mass and recruits less muscle secondary to the surface application (v. subcutaneous or muscle) and the small spread in the drive-stun mode. Furthermore, the animals were noted to have apnea with each TASER exposure. These animals were heavily sedated with drugs known to cause respiratory depression and apnea, and were intubated but not ventilated which would increase the work of breathing. Our study in awake humans showed no apnea with the TASER exposure (with up to 15-second applications). So, it is not likely that the Jauchem, et al. study is relevant in awake humans.

Furthermore, we have conducted an unpublished follow-up study to the breathing study with 18 enrolled subjects in which we duplicated the methodology (including a 15-second continuous discharge from the TASER X26), but added venous blood gas and electrolyte sampling. In this study, there was no significant change in electrolytes or pH. The mean pH change in this study was on the order of -0.001. This would not be considered clinically significant.

In addition, we conducted a similar unpublished experiment with the TASER XREP (a projectile device fired from a shotgun with a similar waveform to the TASER X26). We enrolled 50 subjects. We collected respiratory data, and venous blood gas and electrolytes. In this experiment, a

subset (27) we allowed to have an exposure lasting up to 45 seconds (most self-terminated with a "tap-out" button before that). In this subset (the larger group has not been analyzed), we found the mean change in pH was on the order of -0.01. The one subject who had a 45 second exposure had a pH change of -0.013. This also would not be considered clinically significant.

A criticism of our published study's applicability to this case by one plaintiff expert opinion was that we did not have an exposure to the neck. The exposure location in Mr. Lomax (the posterior neck) is an area heavily invested with subcutaneous fat, musculature (many levels), fascia, ligaments, and bone. The diaphragm, the major muscle of respiration (accounting for 2/3 of the air entering the lungs in a supine adult), is innervated by the phrenic nerves. The phrenic nerve receives its derivation from the ventral rami of C3-5. The C5 fibers may not join the phrenic nerve until it is in the chest. In a large percentage of the population, there exists an accessory phrenic nerve, derived from C5 and C6, and this may not join the main phrenic nerve until the root of the neck or the thorax. This means that disruption of the phrenic nerve in the neck may not paralyze the diaphragm. In addition, the anatomical distance to the phrenic nerve from the exposure location in Mr. Lomax is long, especially when we are discussing the surface application of the current with a drive stun (v. subcutaneous or deeper penetration with the probes). The phrenic nerve, at its most superficial, lies anterior to the anterior scalene muscle just beneath the prevertebral fascia. It is located just posterior to the carotid sheath. This is the antero-lateral neck, not the posterior neck. See Addendum 3 for some anatomic references.

One plaintiff expert opinion indicated that the electrical discharge occurred "just millimeters" away from the nerves which control breathing. A very basic review of anatomy disputes this assertion. Even at its most superficial distance to the skin (at the antero-lateral neck, not the posterior neck), the phrenic nerve is about 2 cm below the skin in an average person (and Mr. Lomax was not average in his subcutaneous depth making this a very conservative estimate). Much of the TASER X26 discharge in the drive stun mode is taken up by the breakdown of the air and skin (both very highly resistive to electrical current). The loud clicking noise heard with exposure in this mode is this air breakdown. The burn marks (compared to a probe exposure) is testimony to the energy wasted on this. This is not a highly effective means of electrical stimulation. Given the anatomic distance and the inefficient electrical energy transfer in this mode, it is very unlikely the phrenic nerve was at all affected by the discharge. The vagus nerve is in

close proximity to the phrenic nerve (the vagus nerve lies in the carotid sheath). We have reasonable evidence that the TASER discharge did not stimulate this nerve since a Fire medic took a pulse shortly after the discharge and found Mr. Lomax to have a pulse greater than 100 (we would expect bradycardia with stimulation of the vagus nerve). The only respiratory muscles then possibly affected by this exposure would be the local accessory muscles, the sternocleidomastoid and possibly the scalenes. And, these are still anterior to the location of the exposure to Mr. Lomax (except for the posterior part of the proximal insertion of the sternocleidomastoid muscle). The posterior neck muscles are not involved in respiration. Additionally, only the ipsilateral side would be affected. The contralateral side would be free to function normally in this type of exposure. And, in the prone position, the contribution to respiration by these accessory muscles is generally small. In my opinion, the 22 seconds (in 1 minute and 47 seconds in the last 5 discharges) in the drive-stun mode would only possibly partially affect the ipsilateral accessory muscles in the neck, and, with their minimal contribution in the prone position, we would expect no effect on respiration.

One plaintiff expert opinion asserts that repeated shocks from the TASER would have damaged the phrenic nerve. We have already addressed the distance from this nerve that the discharges were applied. This expert also has not reviewed the diaphragmatic pacing literature in which patients have their diaphragms paced for long periods of time without damage to the nerve. These time periods are on the order of years. This expert also has failed to understand that there are two phrenic nerves in the neck, one for each side of the diaphragm. Individuals with one diaphragm completely paralyzed from bypass surgery can still have normal oxygenation and ventilation with one diaphragm (they usually will, however, experience dyspnea with exertion). This author also is apparently not aware of the accessory phrenic nerve that is present in much of the population that would make damage to the higher phrenic nerve not likely to lead to diaphragmatic paralysis.

The prone position of Mr. Lomax was criticized in some plaintiff expert opinions. Dr. Chan, et al., in their study ("Restraint Position and Positional Asphyxia", Ann Emerg Med, 30, 1997) found a progressively "restrictive pattern" from sitting to supine to prone to the prone maximal restraint position, but found no evidence of hypoxia or hyercarbia. Without these clinical manifestations, the authors concluded that this "restrictive pattern"

was unimportant. The research of Schmidt et al. supported these conclusions ("The effects of positional restraint on heart rate and oxygen saturation." J Emerg Med, 1999; 17 (5): 777–782). Some criticism of this work stems from the use of healthy volunteers, therefore not replicating the population in whom these deaths are occurring. In the Stratton et al. series, all of the excited delirium deaths (20) occurred in victims in the prone position, but 89% (155/174) of those excited delirium victims that did not have sudden death were also in the prone position. This data makes any association of the deaths to position very difficult. In a series by Paterson et al. of restraint related deaths in non-police custody settings ("Deaths associated with restraint use in health and social care in the U.K. The results of a preliminary survey." J Pyschiatr Ment Health Nurs, 2003; 10: 3-15), in only one of the 12 cases reviewed was the restraint method similar to the "hobble" restraint (an extreme form of prone restraint). Additionally, research in critical care medicine and anesthesiology shows that the prone position may actually improve respiratory status, particularly in compromised patients. There is evidence that this position might be especially beneficial in obese patients. Theories include more uniform lung perfusion in the prone position, and expansion of previously atelectatic areas of lung compressed by the heart and mediastinal structures in the supine position, and increased functional residual capacity. Additionally, the prone position is less likely to lead to aspiration. The seated position, often not possible in actively resisting subjects, is not without risk. Severe respiratory compromise can occur with hyperflexion in this position. The primary benefit of the supine position in patients, especially in uncontrolled emergency settings is better observation of the patient and easier airway accessibility rather than lung function.

The compression is a more debatable issue. Depositions consistently indicated that one security officer had his knee in the sub-scapular area of the back of Mr. Lomax. The degree of this force is debatable. One deposition described it as a "little" pressure. Others made statements that they asked the security officer to remove his knee as they were concerned it might be causing an impairment in his breathing. A study by Dr. Chan, et al. ("Weight Force During Prone Restraint and Respiratory Function", Am J Foren Med and Path, 25:3, 2004) examined the effects of 25 and 50 lbs. of weight on the back while in the "prone maximal restraint position." They found no hypoxia or hypercarbia from either the restraint position (similar to their previous study) or from the restraint position plus the weights. The primary criticisms of this study were the small weight used and the healthy volunteers. Most experts I believe would agree that there is some possibility

of respiratory embarrassment with compression. On transport to the ambulance, Mr. Lomax possibly had a waist restraint in addition to the extremity restraints. I found no description of this procedure, but depending on positioning of the waist restraint, it could have restricted the free movement of the abdomen and therefore impaired his respiration.

Electrolytes and Acidosis

We already referenced the first human effects study by Ho et al. In this study, electrolytes, including potassium, were followed serially after the TASER exposure and no changes were found. Bicarbonate, a surrogate for pH, showed no evidence of a metabolic acidosis. A criticism of this paper was the short duration of exposure. As referenced above, we also conducted a follow-up study to the breathing study with a 15-second continuous exposure and found the mean change in pH was on the order of -0.001. There were no significant electrolyte changes. A similar study of the TASER XREP, with a mean exposure duration of 17 seconds (including one exposure of 45 seconds), showed a mean change in the pH on the order of -0.01. None of these changes would be expected to be clinically significant. See also Exertion below for additional comments on acidosis.

Stress

One plaintiff expert opinion stated that the TASER X26 may induce stress changes that can then have a negative impact on human physiology. There are no studies in the literature on the effects of the TASER on the neurohumoral stress cascade. We have conducted an unpublished study of the neurohumoral response to the TASER X26 versus oleoresin capsicum, commonly known as pepper spray. The LVPD officer was questioned about his other options for use of force. Specifically, he was asked why pepper spray was not used instead of the TASER. Pepper spray has become a relatively uncontroversial use of force (after previous contentious debate). The officer appropriately responded that this use of force was often found to be not effective in highly motivated or intoxicated individuals.

The acute stress response in humans is a neuroendocrine cascade initiated by the hypothalamus. The cascade has two components: the sympathetic-adrenal-medulla axis (SAM), and the hypothalamic-pituitary-adrenal axis (HPA). The SAM axis is responsible for the release of catecholamines, primary epinephrine, from the adrenal medulla chromaffin

cells. These cells contain a pool of catecholamines that are available for immediate release. The HPA axis is responsible for the release of ACTH and β-endorphins. ACTH causes the adrenal cortex to produce glucocorticoids (e.g., cortisol) and mineralocorticoids. β-endorphins modulate pain perception. Cortisol induces an enzyme in the adrenal medulla that is the rate-limiting step in the conversion of norephinephrine to epinephrine. Cortisol also induces gluconeogenesis. Epinephrine release is the key end-result of the neuroendocrine cascade. It produces a number of adaptive physiologic changes in response to stress for a "fight or flight" response, including positive chronotropic and inotropic cardiac effects, increased systemic vascular resistance, increased arterial blood pressure, increased metabolism, and increased thermogenesis. However, it can also produce maladaptive physiologic changes including myocardial ischemia, cardiac dysrythmias, reflex bradycardia (which can cause asystole), pulmonary edema, lactic acidosis, and hyperthermia. Even the adaptive changes can be considered maladaptive when the "fight or flight" response will only serve to cause more harm (as in the case of the excited delirium syndrome).

It has been hypothesized that these maladaptive physiologic changes, as well as the adaptive changes (since a "fight or fight" response is actually counter to what is needed), may be contributory to some in-custody deaths. This may be especially the case when the acute stress response is potentiated by the use of cocaine, amphetamines, PCP or other sympathomimetic stimulants or when psychiatric illness induces a greater perceived threat in the police contact and therefore a greater acute stress response, or when the subject is in a state of excited delirium. Stress has been shown to precipitate acute cardiomyopathy in humans. Recent literature has shown that salivary markers may be able to be used to quantitatively measure the stress response in subjects exposed to stress. The advantage of salivary markers is eliminating the stress of a needle stick to draw serum measures. Salivary alpha-amylase is a validated measure of the SAM axis. Salivary cortisol is a validated measure of the HPA axis. Previous studies have demonstrated that these measures peak 10-20 minutes after the presentation of the noxious stimulus, with alpha-amylase peaking at about 10 minutes, and cortisol at about 20 minutes.

In our unpublished study, we examined the acute stress response to a TASER X26 discharge versus oleoresin capsicum, a commonly used, and generally uncontroversial use of force option. We exposed 10 subjects to a few second spray of oleoresin capsicum to the eyes

(the usual target) and 5 subjects to a standard 5-second TASER X26 exposure with the probes shot into the back from about 7 feet. We measured alpha-salivary amylase and cortisol at baseline, at 10 minutes, 20 minutes, and 1 hour. Our result showed that salivary alpha-amylase increased 173% at 10 minutes with the oleoresin capsicum compared to -8.5% for the TASER. Salivary cortisol increased 89% with the oleoresin capsicum versus 90% with the TASER at 20 minutes. This demonstrates a greater SAM axis response to the oleoresin capsicum and an equivalent HPA axis response.

Mr. Lomax had an elevated neurohumoral stress cascade due to his restraint and struggle, due to the sympathomimetic activity induced by PCP toxicity, and likely due to his excited delirium. Any contribution to the cascade by the TASER X26 exposure, if any, would be negligible in this background. We found that a 5-second burst from the TASER X26 was less than or no different of a "stressor" by the objective measures of salivary alpha-amylase and cortisol than pepper spray.

Exertion Data

In another unpublished study, we examined the effects of a 15-second continuous TASER X26 discharge immediately after a short-duration, maximal intensity exercise regimen. In this study of 44 subjects, we found the mean venous pH at baseline to be 7.37. After the maximal intensity exercise regimen, we found the mean pH to drop to 7.258. After the 15-second continuous TASER discharge, the mean pH remained flat (7.225, not a statistically significant change). The recovery pH returned to baseline (mean pH 7.358). Additionally, lactate had a mean of 1.8 at baseline, increased to 8.7 after the exertion, remained flat after the TASER exposure (mean 9.4, not a statistically significant change), and returned to baseline at recovery. The maximal intensity exercise regimen was brief. It consisted of 30 seconds of push-ups followed by a brief run on the treadmill at 8 mph at an 8% grade. Most study subjects only performed the run for a few minutes. This data suggest that physical struggle to obtain restraint might be more detrimental to subjects who are already acidotic than an application of the TASER.

Temperature Data

We conducted an unpublished study (currently under review by Forensic Science International) of core body temperature in 21 subjects

exposed to a 15-second continuous exposure from the TASER X26. In this study, we found no core body temperature rise with the exposure. Mr. Lomax was sweating profusely and repetitively lifting his shirt. There is no documentation of an initial measured temperature in the provided records except a note by the admitting physician at Valley Hospital Medical Center of "afebrile." This is presumably a temperature obtained after the 40 minutes of ACLS until return to spontaneous circulation in the ED. Therefore a determination of his initial temperature is likely not possible. There is also a later note of a temperature greater than 101 (although he did have pneumonia). Some degree of initial hyperthermia cannot be excluded from these records. Our study does not suggest a contribution by the TASER X26.

Demographic Data

Dr. Strote in his paper "TASER Use in Restraint-Related Deaths" (Prehospital Emergency Care, 10:4, 2006) used web-based search engines to find in-custody deaths in which a TASER was used. In his study, he found that illegal drugs were found in 78% of cases (82% of which were stimulants) and that a specific diagnosis of excited delirium was given in 76%. The autopsy reports listed the TASER as a "potential" cause in 16% of the cases, and as a "contributory cause" in 11% of cases. The author himself notes the limitations that a TASER is frequently used in cases in which the behavior of the suspect already puts him at risk of sudden death, and that temporal association and causality are different and demonstrating a causal relationship to death is "difficult." Additionally, this study only looked at deaths where a TASER was used. It did not look at the broader scope of in-custody death. Dr Ho, et al. ("Deaths in police custody: an 8 month surveillance study", Ann Emerg Med, 2005;46 (suppl):S94) also used an internet search technique to look at all in-custody deaths in a 12 month period. In this study, the TASER was used in 30% of the 162 deaths. In no case was the TASER associated with "immediate" death. In the Stratton case series, a TASER was only used in 28% of the cases (and pepper spray was used in 33% of the cases). In this series, the most associated factors were forceful struggle (100%), stimulant drug use (78%), established natural disease states (including heart muscle fibrosis like Mr. Lomax) (56%), and obesity (56%). In a study by Ross, et al. (Sudden Deaths In Custody, edited by Ross, D. and Chan, T., Humana Press, 2006), a TASER (or other "stun gun") was only used in 21% of cases. In this study, none of the deaths were attributed to the TASER. This data demonstrates that a TASER is used in the

minority of in-custody deaths, and that other factors, many of which Mr. Lomax demonstrated, are more associated with these deaths.

Conclusions About the Cause of Death

The following is my conclusion based on the facts of the case and the data presented above to a reasonable degree of medical certainty:

Mr. Lomax had an acute medical emergency. Whether it was the excited delirium syndrome or the toxic effects of PCP, marijuana, and possibly other non-detected substances, or contributions from both is debatable. My medical opinion is he had the constellation of symptoms, signs, and a pattern of death most consistent with the excited delirium syndrome. What is not debatable is that he needed immediate medical care, and this was not possible without restraint in some manner, even by EMS reports. Even if he had been cooperative, some level of restraint would have been necessary for the protection of EMS personnel given his past violent behavior and his unprovoked battery of the security officer in this case. Mr. Lomax was not being provoked, and indeed, the security officers and the LVPD officer tried to explain that they were only summoning medical help for him. He did not seem to respond to security officers or the LVPD officer in a manner that would show a potential for verbal control, and, indeed, he attacked a security officer unprovoked. Security officers and the LVPD officer had adequately contained him and had a reasonable number of personnel to assume a rapid take-down would be possible. He just proved to be more difficult to restrain. As the Fire Captain stated in his deposition, even "with four officers on him, it was still a battle." In addition, EMS protocols required a change of the restraints. Mr. Lomax, through his drug abuse, created a situation requiring his need for restraint.

The dissociative and anesthetic effect of PCP makes continued resistance against overwhelming force and pain possible. This, combined with its sypathomimetic stimulating features, creates an environment in which a hypersympathetic state, hyperthermia, and acidosis worsen, but the feedback to stop the behavior from worsening is blunted. This is also a classic feature of the excited delirium syndrome. The exact patho-physiological mechanisms involved with this in the excited delirium syndrome have not yet been elucidated but may be related to excess dopamine due to relatively small numbers of and the dysfunctional state of

dopamine transporters as theorized by Dr. Mash. In the case of Mr. Lomax, even with multiple persons attempting restraint, it was difficult to obtain physical control of him.

In my opinion, without the assistance of the TASER X26, the struggle would likely have been more protracted, his deterioration more accelerated, and care even more delayed. In addition, he may have required more restraint to include more compressive restraint that could then have had an effect on his respiration. In his testimony, the LVPD officer said in the previous encounter, in which no TASER was used, they had to "dogpile" him to get him restrained. The LVPD officer testified that other uses of force that might have been employed, in the absence of the TASER, would include the baton, O.C., and the lateral vascular neck restraint (LVNR). We have already seen evidence that O.C. may represent a greater stressor and we know it is not very effective against subjects with altered mental status. This would have likely caused stress with no benefit in this case. In addition, it may have reduced the capability to effectively restrain Mr. Lomax since it would have affected the security officers and the LVPD officer given their need for close contact. The baton, like O.C., is a pain compliance tool. With his altered mental status, it may have not been affective. For the baton to be used as a disabling tool, it would require mechanical impairment, such as broken bones. The baton is well known to have the potential for serious injury. Additionally, with the close proximity of the security officers, the chance for accidental injury is high. The LVNR disrupts blood flow to the brain to induce unconsciousness. It may also work by stimulating the carotid bodies leading to hypotension and unconsciousness. Either mechanism would likely have been precarious in Mr. Lomax who was already in the throes of the excited delirium syndrome.

Why did Mr. Lomax quiet after the last TASER use? First, we know from multiple depositions that he was still breathing and struggling for some time after the last use. So, the timing of the TASER use and his sudden collapse is not exactly coincident. Second, we have at least one deposition that asserted a progressive decline in his resistance over time. The progressive decline is consistent with the progressive worsening of his condition, likely accelerated by the struggle. Third, the last TASER use corresponded with his complete immobilization on the gurney after the last struggle to change the restraints. The dramatic decline after complete immobilization has been reported on before and is characteristic of excited delirium deaths. In the Stratton, et al. series, the cessation of struggle

(quiescent period) before death was seen in all of the excited delirium deaths. In the Ross et al study, 80% of deaths occurred on scene or during transport (60% died on scene). The death occurring shortly after capture is also seen in the veterinary literature ("capture shock" – the use of chemical tranquilizers as opposed to mechanical capture has been found to remedy this problem). There are hypotheses regarding potassium shifts ("period of peril"). There are also theories about catecholamine withdrawal or depletion with subsequent hypotension (and therefore decreased responsiveness) and then cardiovascular collapse.

The temporal association is perilous for drawing cause effect relationships. As discussed above, there is much data to support that the TASER does not cause or worsen the derangements encountered in the excited delirium deaths. If the TASER use is at the end of the struggle, it is then that his metabolic derangements are peaking from the struggle and death is more likely. If this "period of peril" or the catecholamine withdrawal are real phenomena, then if the TASER is what helped stop the struggle, it will be associated temporally as well. The decline of Mr. Lomax was coincident with the change of the restraints, and therefore a last fierce struggle. Since the TASER is often used in exactly the kind of subjects that are high risk for sudden in-custody death, they will often be temporally associated with these deaths. That said, the TASER is not even associated with the majority of in-custody deaths by demographics. In the Ho, et al study, only 30% of in-custody deaths had a TASER used, in the Stratton, et al. series, only 28% had a TASER used, and in the Ross, et al study, only 21% had a TASER used.

In conclusion, the cause of death in this case was likely primarily the excited delirium syndrome precipitated by and coupled with PCP toxicity. A contributing factor would be the prolonged struggle leading to a worsening hypersympathetic state, acidosis, possibly hyperthermia, and electrolyte changes (hyperkalemia from acidosis and rhabdomyolysis or hypokalemia during the "period of peril"). The final cause of death could have been hypokalemia and respiratory arrest, cardiac arrhythmias from electrolyte changes or acidosis, respiratory arrest from acidosis, or cardiovascular collapse from catecholamine withdrawal (or depletion). Any or all of these factors may have played a role in the final event. The degree to which these factors were already occurring before the struggle is not possible to ascertain. However, there is some evidence of his already severely compromised by the descriptions given by the security officers first on

scene. There was likely little to no contribution by his positioning. In fact, this made aspiration less likely and obstruction of his airway by his tongue less likely. There was no contribution by the TASER. This in fact likely assisted in reducing the struggle time. In these cases, early physical control and chemical intervention is the best chance for survival. The veterinary literature on "capture shock" has shown this to be the case. A protracted manual struggle will inevitably worsen the condition. The TASER may actually provide the most rapid and therefore efficacious strategy to obtain control and allow medical intervention in these cases. No specific comment can be made about the effect of the knee in the back or the possible use of the waist restraint. Compressive forces applied to the back or around the abdomen can lead to respiratory compromise. The degree to which this contributed in this case is debatable. It may or may not be a contributing factor depending on the force used with the knee and how the waist restraint was positioned. Additional significant contributing factors would be his obesity, cardiac interstitial fibrosis that suggests chronic heart damage (something that is often found in chronic drug abusers), a pneumonia that may have been aspiration-related due to his mental status, and a hepatitis that might also be drug related.